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Disease-a-Month

Differential Diagnosis of Chest Pain

DAVID DAVIS

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Differential Diagnosis of Chest Pain

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TABLE OF CONTENTS

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ALTHOUGH IT IS AXIOMATIC, the fact that recognition of a disorder demands its consideration in differential diagnosis cannot be stressed too often. It is especially important in dealing with the causes of chest pain. While in over 90% of cases chest pain is due to coronary disease, spinal root compression or a psychogenic disturbance, the many other sources (see table) must be considered if errors are to be avoided. The history must be taken with these sources in mind and the examination is not complete unless, in addition to the usual electrocardiograms, roentgen studies and other indicated procedures are carried out. For example, as chest pain is sometimes an early symptom in carcinoma of the lung, roentgenograms, cytologic studies and bronchoscopy are indicated when the pain or discomfort cannot be decisively attributed to other causes. Early recognition, so important in this condition, depends on "a high index of suspicion."

In the search for the source of chest pain, or any other symptom, it is important not to be misled by initially negative laboratory findings. A single negative roentgenogram of the chest does not exclude the lungs and mediastinum as sources of pain. Just as repeated electrocardiograms are necessary to obtain information concerning coronary disease, pericarditis or pulmonary em-

bolism, so repeated roentgen studies may be necessary to establish the source of pain when it is due to pericarditis and pleuritis, mediastinal emphysema, pulmonary infarction or carcinoma of the lungs.

The urgency for an early or immediate diagnosis is sometimes responsible for an incomplete evaluation of all possible sources. Errors can be avoided if we resist even tentative diagnostic commitments before there has been sufficient time to complete necessary studies and to observe the patient adequately. A diagnosis of coronary disease is still being made more often than warranted. When attention is focused primarily on only the most common sources, the necessary suspicion for the recognition of the less common may be reduced to the point of possibly missing the correct diagnosis.

Another source of error in diagnosis results from failure to remember that two or more sources may coexist in the same patient at one time or in the course of treatment of one of them. Pain due to coronary disease and that due to spinal root compression often coexist. Psychogenic pain may accompany either or both of these. Nor does the presence of coronary disease exclude such other sources as the lungs and mediastinum, the biliary system or upper gastrointestinal tract. When one source is established the possible presence of other sources must still be investigated.

The emphasis in this discussion is placed on the three causes of chest pain that comprise the more than 90%, with extra stress on root compression, only now beginning to be sufficiently recognized. But even in a brief discussion the many less common causes must be included, for one cannot afford to neglect any of them in the differential diagnosis of chest pain.

CHEST PAIN DUE TO MYOCARDIAL ISCHEMIA

PATHOGENESIS.—Myocardial ischemia due to an inadequate coronary circulation is by incidence and morbidity the most important single cause of anterior chest pain. The coronary circulation becomes inadequate when: (1) the caliber of its vessels is reduced in size, (2) heart muscle needs are increased by hypertrophy or (3) blood flow and oxygen capacity become insufficient because of cardiac and extracardiac conditions. Although atherosclerosis, with narrowing and occlusions of the coronary arteries, is by far the most important factor, the

increased circulatory requirements in myocardial hypertrophy secondary to coronary atherosclerosis, hypertension, congestive heart failure or valvular disease play an important part in the development of ischemia. This and other factors influencing

ANTERIOR CHEST PAIN

SOURCES	SYNDROMES
Heart and aorta	Myocardial ischemia Angina pectoris Acute coronary failure Myocardial infarction Acute pericarditis Aortic aneurysms Dissecting, saccular
Thoracic viscera	Pulmonary embolism and infarction Tumors, lungs and mediastinum Mediastinal emphysema Spontaneous pneumothorax Massive collapse of the lungs Acute fibrinous pleurisy
Skeletal, muscular and nervous systems	Spinal root compression Herpes zoster Xiphoid process syndrome Manubriosternal syndrome Cervical rib Costosternal syndrome Pectoral syndrome Coracoid process syndrome
Upper gastrointestinal tract and biliary system	Esophageal lesions Diaphragmatic hiatus hernia Gastric lesions Cholelithiasis and cholecystitis
Mind	Psychosomatic chest pain

coronary blood flow may sometimes be sufficient to cause ischemia even when the main coronary branches show only minor changes.

Depending on the rate of development, degree and local distribution of the atherosclerotic process and the presence or absence of these other factors, three major pain syndromes occur: angina pectoris, coronary failure and myocardial infarction. In angina pectoris the inadequacy of coronary circulation is transient and induced by effort or excitement. In

coronary failure it more often occurs at rest and, although it may be prolonged for hours, the ischemia does not result in gross irreversible muscle changes. In myocardial infarction the changes are permanent.

Although these three distinct manifestations of coronary inadequacy are recognized there are intermediate stages with considerable overlapping of one syndrome by another. Patients with severe angina pectoris and a greatly reduced capacity for effort sometimes have attacks at rest, particularly after meals, and in recumbency. Short attacks lasting from 1 to 5 minutes are often quickly relieved by nitroglycerin. More prolonged attacks may appear later with pain lasting up to hours that is not relieved by nitroglycerin. The short and the prolonged attacks represent varying degrees of coronary failure. They occur in patients with or without angina pectoris. They represent a somewhat more advanced stage of coronary inadequacy and are often the precursors of myocardial infarction.

An understanding of the many factors that play a role in the production of myocardial ischemia is important for diagnosis as well as management. Largely as a result of studies by the injection-dissection technic of Schlesinger (1), the pathologic physiology of these syndromes has been clarified. Normal hearts with no appreciable narrowing of the coronary arteries do not have intercoronary anastomosis of a caliber larger than 40μ. Much finer communications do occur and watery solutions injected into one coronary artery readily reach other coronary vessels. Blood, however, will not flow through anastomoses of this low caliber and when a coronary vessel that is not appreciably narrowed is suddenly occluded by a thrombus, myocardial infarction occurs. On the other hand, when coronary artery narrowing and occlusion occur slowly, compensatory anastomotic circulation with intercoronary communications of a larger caliber are induced and these may be sufficient to prevent infarcts or attacks of pain even after extensive changes have occurred.

In 1940 Blumgart, Schlesinger and Davis (2) noted that patients with long-standing angina pectoris usually have marked narrowing or occlusions in at least 2 main coronary branches and that sometimes occlusions occur in all 3 without gross infarction. The role of myocardial hypertrophy in the production of the ischemia was also stressed by Davis and Klainer (3), who found an extreme degree of coronary dis-

ease usually involving 2 main coronary arteries in 95% of normotensive patients with angina in contrast to an incidence of only 39% in those with hypertension and cardiac hypertrophy. More recently Zoll and associates (4) reported findings in 177 patients with angina pectoris. Although atherosclerosis played a role in 90% of their cases, it was solely the cause of coronary inadequacy in only 16%. Coronary disease plus hypertensive heart disease were involved in 61%; valvular or hypertensive disease alone or valvular disease with coronary disease was responsible for the ischemia in the remaining group. Other anatomic factors in angina pectoris that interfere with coronary circulation are constriction of the coronary ostia in syphilitic aortitis, calcific or rheumatic aortic stenosis and mitral stenosis. In angina pectoris the work of the heart is not only temporarily increased by exertion, emotion and cold, but constantly by the load of hypertension, valvular insufficiency and stenosis or hyperthyroidism when present. It is also increased in tachycardias, infections, pulmonary disease and anemia, but here, in addition, there is interference with coronary flow, oxygen capacity or both. Because of the high incidence of coronary occlusions in patients with angina pectoris we may assume that a sudden narrowing of the coronary bed by a silent occlusion frequently precedes the inception of the syndrome and that a similar occurrence is often responsible for a sudden progression of the disease after a regular pattern has developed.

In acute coronary failure primarily due to atherosclerosis, the changes are the same or perhaps more extensive than they are in angina pectoris. Some attacks of chest pain in this condition are associated with an occlusion without infarction. In 11 cases at necropsy reported by Freedberg and associates (5), there were 2 to 6 old occlusions in 6 patients, marked narrowing of the main branches in 3, and no significant changes in the remaining 2. Significantly, the hearts were appreciably hypertrophied in 8 of the 11, 5 weighing above 600 Gm. Five of the 11 showed some gross fibrosis and all 11 showed microscopic infarction or fibrosis. In the 2 patients who showed little or no coronary changes, the mechanism of the attack in one patient could be attributed to paroxysmal nodal tachycardia.

More often than in angina pectoris, attacks of acute coronary failure also occur as a result of conditions that interfere

with coronary flow or oxygen capacity, such as tachycardias, anemia, valvular disease, congestive heart failure, pulmonary disease, anesthesia, a fall in blood pressure or shock-like states, carbon monoxide poisoning and pulmonary embolism. In congestive heart failure a combination of factors such as coronary sclerosis, myocardial hypertrophy, the load of an increased blood volume and a diminished oxygen saturation may all play a role in the production of the attack.

Myocardial infarction takes place when the total circulation to a given area of muscle falls below its minimal requirement. Sometimes it occurs when a main coronary vessel becomes occluded rapidly before anastomotic circulation has developed but most often it occurs when the total circulation including the anastomotic circulation finally becomes insufficient for a given area of muscle. The same precipitating circumstances that induce attacks of angina pectoris or coronary failure may initiate an attack but most often attacks occur spontaneously.

Angina Pectoris

The term "angina pectoris" was introduced by Heberden (6) in 1768 to call attention to a "disorder of the breast" associated with a "sense of strangling and anxiety." The diagnostic characteristics of the syndrome were tersely described as follows: "They who are afflicted with it are seized while they are walking (more especially if it be up hill, and soon after eating), with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or continue; but the moment they stand still, all this uneasiness vanishes."

When the history unmistakably reveals these essential features: the sudden onset, the breast location, precipitation by exertion, short duration and quick relief by rest, the diagnosis is seldom in doubt and exercise tests under standard conditions will regularly confirm it. When these clinical features are not all present, particularly the close relation to effort, the short duration or the immediate relief by rest, the diagnosis is in doubt and more often in such patients it can be shown by exercise tests and clinical course that angina pectoris is not present and that the pain is due to such other conditions as root compression, diaphragmatic hernia, biliary disease, the xiphoid process syndrome or psychoneurosis. Obviously there

are pitfalls in each group. A few patients who appear to present a typical picture do not have angina pectoris; some with an atypical picture do have it. A common pitfall is an unreliable history where the patient gives a misleading story of the relation of exertion, or lack of it, to the attack. Another is in the case of psychogenic pain, where the patient unconsciously presents a "typical" but simulated picture of angina pectoris based on a previous knowledge of the disease. In rare instances spinal root compression produces chest pain which at first fulfills all the criteria of angina pectoris.

Atypical examples of true angina pectoris may occur at the inception of the illness before the syndrome has developed its characteristic regularity and often in a late stage shortly before the development of coronary failure or myocardial infarction. The relation of the pain to exertion may be present early, but it may not be as consistent then as when a regular pattern has developed; also attacks may occur at rest, throwing further doubt on the exact relation of pain to exertion.

CHARACTER OF PAIN.—Sometimes the pain in angina pectoris is vague and difficult to describe, as Riseman maintains (7). An awareness of this quality can be helpful in diagnosis. More often, in the author's experience, the pain has definite characteristics that are clearly imparted when the patient is capable of a good description. The essential feature of the pain was first pointed out by Heberden as a "sense of strangling" and for this reason he termed it angina pectoris. Most often the patient immediately recognizes that the distress is an unusual one, a feeling that he has not experienced before, and the most common element of the discomfort is this strangulation, pressure or constriction. Such terms as a feeling of tightness, a weight sensation, a squeezing, a vise-like constriction, a choking, a fullness or expanding discomfort are regularly volunteered. Less often the discomfort is described as a dull ache, a boring or burning pain, and only rarely is it said to be sharp.

When the pain is described as a burning sensation it is sometimes confused with esophageal spasm secondary to anxiety, peptic ulcer or hiatus hernia. Not infrequently the pain resembles the distress of gaseous distention and the patient tries to belch in order to obtain relief.

Although the discomfort in angina pectoris is usually moderately severe, on occasions it may be mild and when mild

it may only slow the patient's pace rather than force him to stop. Occasionally it continues for a few minutes and finally disappears as the patient continues at the reduced pace. Severe attacks are frequently associated with a fear of impending doom, accompanied by a peculiar breathing difficulty, pallor and sweating. Such attacks seldom simulate any other disease.

LOCATION AND RADIATION.—The chest pain in angina pectoris may be located anywhere from the one anterior axillary line to the other, from the suprasternal notch to the epigastrium. Most commonly, however, it is located over the middle and upper area of the sternum. Less common sites are the precordium, areas just below the clavicles on either side, any part of the upper extremity, the epigastrium, throat, jaws or teeth. When attacks of pain in the throat, jaws or teeth are closely related to effort and quickly relieved by rest and nitroglycerin, they usually represent angina pectoris even in the absence of chest pain. Pain localized sharply to one small area at the apex or between the apex and the left costal margin, or any other localized part of the chest wall, is usually not angina pectoris. Most often pain so localized is psychogenic in origin. Pain below the clavicles and over the lateral aspects of the chest wall, particularly when associated with shoulder girdle pain, is more frequently due to lower cervical root compression. Constricting substernal pain is not in itself diagnostic of angina pectoris for it is seen in several other conditions and is particularly common in root disease.

In approximately 50% of the cases the pain in angina pectoris radiates from the substernal location, most commonly to the left shoulder and inner aspect of the left arm. It may radiate to both extremities, only to the right upper extremity, the jaw, throat or scapular regions posteriorly. Occasionally the pain starts in the shoulder, inner aspect of the forearm, left wrist or tips of the ring and little fingers and radiates up the extremity to the substernal region.

It has been stated that radiation from outer aspects of the chest, the shoulder and upper extremities to the substernal region is characteristic of angina pectoris, but this type of radiation is also seen with root compression, where the pain may start in the extremity, shoulder or back and radiate to the anterior chest wall. Radiation from the chest down the outer aspect of the extremities is more characteristic of root disease than of angina pectoris.

RELATION TO EXERTION OR EXCITEMENT.—The relation to exertion and cardiac effort is the most important diagnostic feature of the syndrome. If the pain is induced by walking slowly, it is more readily induced by walking rapidly; if it is induced by walking on level ground, it will more likely be induced by walking uphill or climbing stairs. When there is any doubt as to the relation, the patient should be observed directly in the course of activity or by exercise tolerance tests. As noted, there are patients with angina pectoris who at one stage, usually at the inception of the syndrome or shortly before impending coronary failure, show discrepancies between a given degree of cardiac effort and chest pain. An attack may occur with little effort on one occasion and not after considerably more exertion on another, causing confusion and raising doubt as to the diagnosis. This stage, however, usually does not last long. When such discrepancies occur one should reserve judgment until the patient can be observed over a period of weeks or longer. Even after a regular pattern of angina pectoris has developed and is maintained, a remission may occur and failure to induce attacks even after exercise tests does not exclude it. Attacks may be precipitated by anger, fear or any pleasurable excitement such as the discovery of an exceptional hand in cards.

ATTACKS AT REST.—Except for occasional remissions, patients with angina pectoris usually remain at a given capacity level for long periods without much variation. Exercise tests under standard conditions show a remarkably constant capacity when repeated over a period of months. Even after this pattern is established patients with a more limited capacity may have an attack while at rest. Such attacks at rest, however, are infrequent unless acute coronary changes are taking place and the patient is beginning to have bouts of acute coronary failure. When attacks of chest pain at rest are numerous over a period of months and there has been no decrease in the patient's capacity for effort without pain, the diagnosis of angina pectoris should be questioned. As noted, attacks at rest are not uncommon at the inception of angina pectoris or just before the onset of coronary failure or infarction, but they are uncommon once a regular pattern has developed and the patient's capacity for effort is not greatly impaired.

SUDDEN ONSET, SHORT DURATION, AND RELIEF WITH REST AND NITROGLYCERIN.—The attack of angina occurs quite suddenly

in the course of exertion and most often, within a few seconds of the first discomfort, the distress mounts rapidly in severity and forces the patient to stop or slow down. It is the exception when the pain increases gradually so that the patient may continue to walk a block or more before he is finally forced to slow down or stop. Pain that takes over 5 minutes before reaching its maximum if the exertion is not curtailed is not likely to be of coronary origin.

Most often, once the attack has started the duration of pain is short. Riseman and Brown (8) found that 90% of attacks on exertion last less than 3 minutes although the patient usually overestimates the duration. If the patient rests when the attack begins, the pain subsides in less than 1 or 2 minutes depending on the severity to start with. This immediate subsidence of the attack by rest is one of the important diagnostic characteristics of the syndrome. Subsidence with rest is even more dramatic after nitroglycerin and this fact is of diagnostic value. Root pain and psychosomatic pain usually last much longer and subside more gradually.

OBJECTIVE FINDINGS.—The physical and laboratory findings in angina pectoris are not of great value in diagnosis. Approximately half the patients show hypertension, some cardiac enlargement or electrocardiographic changes. Angina is infrequent in women under the age of 50 without hypertension or diabetes but it does occur, and chest pain in these patients requires the same critical evaluation.

EXERCISE TOLERANCE TESTS.—Two tests that have been popular in the past two decades give information concerning the presence or absence of angina pectoris or myocardial ischemia. The standardized exercise test of Riseman and Stern (9) has been used successfully at the Beth Israel Hospital in Boston since 1932. In this test, attacks of angina are induced by having the subject mount and descend a two-step staircase in a cold room, 45-55 F., or while holding an ice cube in one hand (10). The test is positive when the patient is forced to stop because of the onset of pain typical of angina pectoris and identical with the pain experienced in spontaneous attacks. Details of the attack can be observed directly: the sudden onset, the patient often going only 2 to 6 trips before being forced to stop, the location, radiation and short duration. The amount of exercise required to induce the attack is later checked by a second test and, unless there has been a basic change in the

patient's clinical condition, an exacerbation or remission, typical attacks will be induced after a similar amount of exercise provided the same standard conditions are carried out as to temperature or ice cube, meals and medication. With this test, false negative and false positive results are rare. Riseman does not recommend the test as a routine office procedure because of the unpredictable course of patients with angina pectoris and the possibility that exercise may induce a fatal attack. Yet in his experience in the past 23 years with over 10,000 tests on many hundreds of patients untoward effects have occurred in only 5 instances.

The Master test, which has gained considerable popularity, is an indirect test to determine the presence or absence of exercise-induced myocardial ischemia as evidenced by the development of electrocardiographic changes (11, 12, 13). Three standard and one apical precordial lead are obtained while the patient is at rest, and with the cables still attached to the subject but not to the machine, the patient mounts and descends the two-step staircase. Exercises are carried out for 1½ minutes, the number of trips performed being regulated according to age, sex and weight of the patient. Immediately after exercise an electrocardiogram with these four leads is again taken and compared with the tracing at rest. A positive result is indicated by changes in one or more leads consisting of a significant ST depression with or without a tendency of the T wave to be inverted. If the result is negative it can be repeated with the patient performing twice as many trips in twice the time. This test, unlike the Riseman test, which gives evidence as to the presence or absence of angina pectoris, discloses only the presence of ischemic changes after exercise. Such changes do not signify angina pectoris and their absence does not exclude it. Useful information, however, is obtained. Patients with such ischemic changes probably have significant underlying coronary disease. If they do not have angina pectoris or coronary failure they are more likely to develop it.

For many years I have combined both tests. The combined exercise test is carried out before a meal on a patient who has had no attacks in the previous 24 hours. It is not carried out if capacity for effort is known to be greatly limited, if there has been any recent exacerbation of symptoms or if attacks have occurred at rest. An electrocardiogram is taken at rest. With the electrodes attached to the extremities, the patient is

exercised with an ice cube held in the left hand. Immediately after the development of an attack the patient is put back on the table and the cable is attached to the electrodes. This requires about 1 minute. Electrocardiograms are then taken at intervals up to 15 minutes. The patient who does not develop an attack is allowed to continue until some dyspnea has developed, after which electrocardiograms are taken in the same manner.

Acute Coronary Failure (Coronary Insufficiency)

Prolonged anterior chest pain at rest or in recumbency in patients with angina pectoris, formerly referred to as status anginosus or angina decubitus, has long been recognized as a serious manifestation of coronary disease. Because such attacks represent a distinctive intermediate stage between angina pectoris and myocardial infarction, because they occur also in coronary disease without angina pectoris, and in several other cardiac and extracardiac conditions where coronary inadequacy is induced by a diminished blood flow or reduced oxygen capacity, there has been need for a more comprehensive and descriptive designation such as "acute coronary failure" (Blumgart, *et al.*) (14) or "acute coronary insufficiency" (Master, *et al.*) (15).

Clinical forms.—When the syndrome occurs in arteriosclerotic and hypertensive heart disease with progressive narrowing of the coronary bed, it appears in two forms: (1) a succession of recurring attacks at rest, often with increasing severity and at successively shorter intervals over a relatively short period of days, weeks or months, and culmination in an attack of myocardial infarction and (2) isolated attacks with intervals of months or years before the onset of myocardial infarction or death. In Freedberg's report of 11 cases at necropsy, the intervals before death in 5 patients were 2, 3½, 5, 9 and 19½ years. Some attacks, particularly the isolated ones, are probably due to an occlusion, without gross infarction, of a major or minor coronary branch. Most attacks develop spontaneously but some appear to follow or are precipitated by excessive physical exertion, emotional stress, hypertensive crises or overindulgence in food or alcohol.

Attacks of chest pain in chronic rheumatic and syphilitic heart disease fall into this category and are usually due to deformities of the aortic and mitral valves which interfere

with blood flow through the ostia of the coronary arteries. Here muscle hypertrophy and increased cardiac work secondary to valvular disease play an important role. In congenital heart disease, attacks of acute coronary failure may be due to several factors: mechanical interference with coronary flow, impaired oxygen saturation, pulmonary hypertension and cor pulmonale. In a form of primary pulmonary hypertension occurring chiefly in young adults, attacks of dyspnea, precordial pain and syncope are often precipitated by exertion (16). Diagnosis is based on the presence of clinical, electrocardiographic and roentgen evidence of cor pulmonale without other cardiac abnormalities. When any form of heart disease is complicated by congestive heart failure, coronary flow and oxygen saturation are impaired, and with an expanded blood volume cardiac work is increased further.

Attacks of acute coronary failure also occur in the absence of heart disease, in other conditions where coronary flow or oxygen saturation is decreased. A fall in blood pressure such as occurs in shock, hemorrhage, spinal anesthesia, operations or trauma may induce an attack. In anemia, hemorrhage, anesthesia, asphyxia, pulmonary embolism, chronic pulmonary disease, severe bronchial asthma or carbon monoxide poisoning, it is associated with a reduction in oxygen capacity. In all these conditions a combination of several factors is usually involved.

CHARACTER OF PAIN.—The pain in this syndrome is more severe and persistent than in angina pectoris but usually not as overwhelming as it often is in myocardial infarction. The oppressive, constricting pressure quality, the location and radiation are the same in all three. Attacks vary greatly in duration, lasting anywhere from a few minutes to an hour and sometimes for several hours. The intensity of the pain often persists at a given level without too much variation and the patient usually remains motionless during the attack. More often than in angina pectoris it is accompanied by weakness, apprehension and sweating, and occasionally attacks are accompanied by cyanosis and restlessness. The blood pressure is usually sustained. Nitroglycerin is seldom effective and morphine is required to give substantial relief. In a very small group of patients, when attacks are severe and the pain prolonged for hours, a shock-like state and occasionally pulmonary edema will develop.

DIFFERENTIAL DIAGNOSIS.—When attacks of prolonged chest pain occur in the course of increasingly severe angina pectoris, a diagnosis of coronary failure or myocardial infarction is naturally given first consideration. To a lesser degree this applies to prolonged attacks in chronic rheumatic, syphilitic or congenital heart disease. In patients without angina pectoris, valvular heart disease or a history of past infarction, differential diagnosis may be more difficult and several causes of chest pain will have to be given consideration. The presence or absence of extracardiac conditions that result in a diminished coronary blood flow or a reduced oxygen saturation must be determined. When chest pain occurs in the course of congestive heart failure, coronary failure must be differentiated not only from myocardial infarction but from pulmonary embolism.

Differential diagnosis between acute coronary failure and myocardial infarction is seldom difficult. The severity, character and location of the pain may be the same in both conditions but in the former, signs of myocardial necrosis such as fever, leukocytosis, an elevated sedimentation rate or an increase in serum transaminase are absent and, more important, the progressive electrocardiographic changes of infarction do not occur. The electrocardiogram may be entirely negative or show only minor changes such as occur during attacks of angina pectoris, which return to normal or the pattern present before the attack in hours or days. Such transient changes usually confirm the diagnosis.

Differentiation from anterior chest pain of spinal root origin may present a problem. Root compression causes attacks of prolonged chest pain at rest with the same character, location and radiation as in coronary failure. Frequently attacks appear to be related to exertion. Often they are associated with a peculiar respiratory distress. This combination at first strongly suggests coronary disease. Such attacks, however, are usually related to certain postures and influenced by movements of the head, neck and trunk. When precipitated by exertion the pain is related to body movements rather than to cardiac work. Physical examination will reveal tenderness over the involved area of the spine and frequently pressure applied to the spine will reproduce the chest pain. Tenderness over the chest wall is regularly present. The therapeutic response to traction will usually confirm the diagnosis. The various characteristics of root chest pain are dealt with more fully in a subsequent section.

Chest pain of psychogenic origin may also occur in the form of attacks at rest or in recumbency, and in location and character closely simulate attacks of coronary failure. However, although prolonged, the pain is usually not sustained with the same intensity, and onset and offset are likely to be more gradual. When seen in an attack the patient can often be diverted by conversation. Other manifestations of a psychoneurosis may be apparent or elicited but often a suspicion that an attack is of psychogenic origin can be confirmed only after prolonged observation.

Acute coronary failure must also be differentiated from benign idiopathic pericarditis, mediastinal emphysema, tumors of the lungs and mediastinum, gallbladder disease and diaphragmatic hernia. The diagnostic aspect of these syndromes are to be considered later.

Acute Myocardial Infarction

Because of its high incidence and the well-known fact that its symptoms can sometimes be mild, there has been a tendency to make this diagnosis more often than warranted. Too often old and very minor electrocardiographic changes have been erroneously interpreted as evidence of infarction. When infarction has occurred, electrocardiograms taken at frequent intervals will regularly show diagnostic changes. High lateral or posterior wall infarction may produce only slight changes in the conventional 12-lead electrocardiogram, but extra leads taken over the left upper chest or high in the axilla will often give the additional information necessary for a positive diagnosis. Extensive infarction is regularly attended by fever, leukocytosis, an increased sedimentation rate and an elevated serum glutamic oxalacetic transaminase level within 12 to 48 hours after onset. When the area involved is small, fever may be absent and leukocytosis slight or absent. The sedimentation rate, however, is more likely to show an elevation and the transaminase values are more regularly elevated. Depending on the degree of necrosis, levels for the latter rise from a normal of 8 to 40 units per millimeter to from 70 to 600 units in 12 hours to 6 days (17, 18, 19).

The pain in acute myocardial infarction is in character, location and radiation similar to that of angina pectoris or coronary failure. The attacks, however with exceptions, are of

longer duration and of greater severity. Most often the pain lasts for more than a half hour and frequently for several hours depending partly on the size of the infarct. In most instances the diagnosis can be made on the basis of the symptom picture and confirmed by the electrocardiogram. In a small group, however, the diagnosis is at first not so obvious. Pain may not be the typically severe kind and its duration may be relatively shorter. In rare instances pain may be absent and the onset of the attack may be characterized only by the development of paroxysmal dyspnea, sudden weakness, syncope or collapse. Painless infarction may also occur in the course of tachycardia, hemorrhage, congestive heart failure or after a fall in blood pressure from any cause. It should be considered when there is a sudden onset of acute pulmonary edema, a sudden aggravation of signs and symptoms in the course of congestive failure or the appearance of arrhythmias, particularly ventricular tachycardia or heart block with or without chest pain.

The symptom picture in acute myocardial infarction depends largely on the size of the infarct. When the infarct is large, in addition to pain there may be dyspnea, weakness, nausea, vomiting, excessive perspiration, cold moist extremities or collapse. Physical examination may show pallor, basal rales, cyanosis, distant heart sounds, gallop rhythm, a friction rub or arrhythmias. The blood pressure usually falls soon after the cessation of pain; less often it falls more slowly in the next 24-48 hours, and sometimes only gradually in the course of several days. The gallop rhythm is best heard 1 or 2 cm. within the apex to the left sternal border and is either protodiastolic or presystolic in time. A pericardial friction rub is noted in 10-20% of cases. It may be audible a few hours after the attack but more often it is present on the second or third day. It is best heard just within the apex or along the left border of the sternum.

DIFFERENTIAL DIAGNOSIS.—The electrocardiographic findings will regularly establish the diagnosis of myocardial infarction. Before these are available a tentative diagnosis will depend on the severity, the location of pain and other characteristics of the attack. Pain of moderate severity, localized to the mid- or upper sternal region, with or without radiation and not associated with appreciable weakness, must be differentiated from the more common causes of chest pain of this magnitude, namely, acute coronary failure, benign idiopathic pericarditis, pulmonary embolism, spinal root compression and psychosomatic pain.

Root compression may cause an attack of acute anterior chest pain that closely simulates that of myocardial infarction. The objective signs of root compression noted in the discussion on acute coronary failure will be present and establish the diagnosis.

In acute benign idiopathic pericarditis, the pain may be localized to the midsternal area and closely resemble the constricting pain of myocardial infarction. It may be influenced by bodily positions, deep breathing and cough. Fever, leukocytosis and an elevated sedimentation rate usually occur with the onset of pain in contrast to the more delayed appearance of these changes following myocardial infarction. In contrast to infarction, the serum transaminase level is usually normal. A friction rub occurs frequently and when present is more widespread than in myocardial infarction and likewise occurs at the beginning of the illness in contrast to the delayed onset following myocardial infarction. The electrocardiogram may show elevation of the RST intervals in the precordial and standard leads. Roentgen examination of the chest will often reveal at least a small amount of pleural effusion.

In pulmonary embolism chest pain may be absent or vary from mild to very severe depending on the size of the embolism. When a main branch of the pulmonary artery is occluded the attack may closely simulate acute myocardial infarction. If the chest pain is accompanied by cough and hemoptysis the diagnosis of embolism is more obvious but often these symptoms are absent. The electrocardiogram, if taken shortly after the onset of pain, may be of diagnostic value. Pulmonary embolism may cause an acute cor pulmonale with changes that, because of a deep Q wave in Lead III, superficially resemble those of posterior wall infarction. Such findings as a deep S in lead I, inverted T waves in the right chest leads V_1 to V_3, clockwise rotation indicated by a shift to the left of the transitional QRS in the chest leads, transient right bundle branch block, a late R in aVr and the absence of large Q waves in lead aVf will readily differentiate it from infarction. Furthermore, the electrocardiographic changes due to acute cor pulmonale usually return to normal quickly. A comparison of the transaminase levels in both conditions will also be helpful. They are usually normal in pulmonary embolism but, when elevated, values are much lower and the peak level occurs much later (3 to 6 days) than in myocardial infarction.

Unusually severe attacks of pain with a very abrupt onset must be differentiated from dissecting aneurysm of the aorta. In this condition the pain is likely to be more widespread and to have a tearing, lancinating quality. It usually persists for hours despite Demerol® and morphine. There is nearly always a history of hypertension and, most important, the blood pressure is likely to be sustained throughout the attack in contrast to the usual fall of blood pressure in severe attacks of infarction. Examination of the heart is not remarkable and the electrocardiographic changes seen in infarction are absent.

Chest pain localized to the lower sternum or epigastrium, particularly when associated with nausea, vomiting or belching, must be differentiated from gallbladder disease, a diaphragmatic hernia, acute gastritis or an active gastric or duodenal ulcer. Lower anterior chest pain, often associated with collapse but usually accompanied by a predominance of abdominal symptoms, also occurs in acute abdominal conditions such as a perforated ulcer, acute pancreatitis, intestinal obstruction and, rarely, acute appendicitis or renal colic. As minor electrocardiographic changes may be associated with these conditions one should be careful not to misinterpret the changes. A careful examination of the abdomen as well as the chest combined with indicated roentgen studies of the gallbladder and the gastrointestinal and urinary tracts, and serum amylase studies will help to establish the correct diagnosis.

BENIGN IDIOPATHIC PERICARDITIS

Pericarditis of any origin may cause anterior chest pain. The specific etiologic varieties such as rheumatic, tuberculous, bacterial, uremic, traumatic or malignant will most often be recognized by the presence of the underlying disease.

Acute nonspecific pericarditis, on the other hand, frequently appears in otherwise healthy subjects and the attack may closely simulate other causes of chest pain. The pain in pericarditis is believed to arise from irritation of the lower parietal pericardium and the contiguous mediastinal, costal or diaphragmatic pleura (20). The pain may be sharp and pleuritic or dull and oppressive. It has been described as a "ballooning out" feeling or sensation of intrathoracic pressure. It may be localized to the precordium or the substernal region or spread over

a wide area of the chest. Radiation to the interscapular region posteriorly, neck, epigastrium, shoulders or arms and down to the fingers is common. The pain is often aggravated by deep breathing, coughing, laughing, swallowing or movements of the trunk, and in this respect it simulates the pain of a spinal root compression. It may be accompanied by dysphagia, dyspnea and hiccough.

A friction rub is common but not invariably present. When it occurs it is often loud and more widespread than the rub following myocardial infarction. It is usually present from the onset of the pain and it may persist for 7-10 days. A low-grade fever is common but may be absent. Pericardial effusion may or may not occur. Some degree of pleural effusion, however, is common and when the amount is small the roentgenogram gives this information. Fever, leukocytosis and elevation of the sedimentation rate occur much earlier with benign idiopathic pericarditis than with myocardial infarction and the transaminase levels are normal or only slightly elevated in contrast to the striking elevation in the latter. The white blood count is usually above 10,000. Paracentesis usually reveals bloody fluid. Pericarditis is sometimes accompanied by acute cardiac dilatation which rapidly returns to normal in a few days.

Electrocardiographic changes occur frequently in the early stages and they are best observed in the precordial leads as an elevation of the RST segment. The RST segment is also elevated in one or more standard leads without reciprocal ST depressions such as occur in acute myocardial infarction, where an elevation in lead I is associated with a depression in lead III or leads II and III. Here also the elevated segment is usually either concave upward or horizontal to the top of the T wave in contrast to an upward convexity in acute myocardial infarction. Inverted T waves develop after the ST changes return to the base line. Abnormal Q waves characteristic of infarction do not occur in pericarditis.

Some difficulty occurs when myocardial infarction is complicated by acute pericarditis. The combination is usually characterized by simultaneous RST elevations in the standard leads plus the presence of Q waves in leads I, aVl and precordial leads, or leads II, III and aVf. As the infarct evolves, the reciprocal changes characteristic of infarction will occur and it will be apparent that both conditions were present.

DISSECTING AND SACCULAR ANEURYSMS OF THE AORTA

Dissecting aneurysm of the aorta occurs in hypertensive males between the ages of 40 and 70, rarely in young adults. The intimal tear usually takes place a few centimeters above the aortic valve and near the subclavian artery but it may occur in any part of the thoracic aorta. Attacks appear to be precipitated by great physical exertion. The violence of the attack resembles that of acute myocardial infarction but the pain is likely to be more abrupt in onset, sharper and more tearing in quality and usually more widespread than in acute myocardial infarction.

Radiation of pain from the substernal region occurs to the head and neck, back of chest, abdomen, lumbar and pelvic regions, and upper and lower extremities depending on the site of the aneurysm and the extension of the process. Wherever the dissection interrupts arterial branches of the aorta that supply various parts of the body, symptoms are produced. The result is a variety of bizarre and far-flung symptoms such as temporary loss of vision, numbness, paralysis of the upper or lower extremities or hematuria.

When the rupture occurs close to the aortic ring, signs of aortic insufficiency may appear. Pulsations over the subclavian or iliac arteries may be diminished or absent and the radial pulses may show striking differences. Pleural effusion may develop. A relative anemia due to extravasation of blood may occur and the icteric index becomes elevated. The patient is usually in shock and despite this the blood pressure remains elevated in contrast to the usual fall in myocardial infarction. There may be differences in blood pressure in the two arms and blood pressure in the lower extremities may be comparatively diminished. Dysphagia due to pressure of the false sac on the esophagus, when it occurs, also helps to differentiate the attack from acute myocardial infarction.

Roentgen examination sometimes shows a progressive widening of the aorta, which appears irregular and distorted by shadows, and successive films may give evidence of extension. Occasionally, the electrocardiogram may show nonspecific RST and T wave changes but the characteristic progressive features of acute myocardial infarction are regularly absent—one of the most important differentiating features.

Syphilitic saccular aneurysms of the ascending aorta arising

between the aortic valve and the innominate artery may reach considerable size. They produce few symptoms but striking physical signs. The heart is displaced downward and to the left, and the sac takes the position above and to the right. Visible pulsations, a palpable systolic thrill and diastolic shock, dullness and a rough systolic murmur may be noted over the upper right interspaces, the suprasternal notch and the manubrium. Pressure of the sac on the innominate artery may produce diminished pulsations over the right carotid, subclavian, brachial and radial arteries with a lowered blood pressure in the right arm.

Aneurysms arising from the transverse arch produce a variety of symptoms. Compression of the right bronchus, trachea and lungs will produce cough and dyspnea; of the recurrent laryngeal nerve, a brassy cough and hoarseness; of the esophagus, dysphagia. Compression of the superior vena cava will cause cyanosis with or without venous engorgement and edema of the face, neck and upper trunk; unilateral sweating may result from pressure on sympathetic ganglia.

In addition to these symptoms, anterior chest pain is common. It may appear as a constant substernal discomfort with pain referred to the neck, shoulder and back of the chest. At times mild, it may become severe and, when there is bone erosion of the clavicle, sternum or ribs, it may be almost intolerable. It is often intensified or relieved by certain positions. The diagnosis of a saccular aneurysm is based on evidence of syphilis, demonstration of a mediastinal tumor that is expansile on physical or roentgen examination, and particularly by angiography, which will often differentiate it from a neoplasm, when the sac is not expansile.

PULMONARY EMBOLISM AND INFARCTION

Bed rest and immobilization, operative procedures, local phlebitis, congestive heart failure, auricular fibrillation, mitral stenosis and bacterial endocarditis are conditions that predispose to pulmonary embolism. When the source is the deep veins of the legs, local signs may or may not be present but venography will usually establish its presence.

Most patients with pulmonary emboli do not have significant chest pain. Small emboli usually produce transient episodes of

mild symptoms such as unexplained fever, weakness, slight dyspnea, hyperpnea, tachycardia, or transient auricular fibrillation, with little or no chest discomfort. Larger emboli may cause: (1) gross areas of pulmonary infarction and (2) an acute cor pulmonale. Gross areas of pulmonary infarction that involve the pleura are usually accompanied by sharp pleuritic pain, aggravated by inspiration. Cough, bloody sputum, fever, leukocytosis, physical and roentgenologic signs of consolidation and pleural effusion may or may not occur. Cor pulmonale is usually accompanied by severe oppressive substernal pain (coronary failure) and signs of acute right ventricular strain or failure, such as dyspnea, pulmonary edema, an accentuated P-2, engorgement of the neck veins, and electrocardiographic changes. The roentgenogram may show dilatation of the right ventricle and pulmonary artery. Such changes are more regularly present when a main branch of the pulmonary artery is occluded. Massive emboli that involve the main pulmonary vessels may also produce shock, mental symptoms, convulsions or sudden death.

When the chest pain is sharp and pleuritic in character and is aggravated by deep breathing, it is easily differentiated from the pain of myocardial infarction. When it is oppressive and confined to the sternal region, differential diagnosis may be difficult. In embolism the pain usually does not radiate to the upper extremities as it often does in myocardial infarction. Serial determinations of serum glutamic oxalacetic transaminase may aid in differentiating the two conditions. In myocardial infarction levels are high and start within 12 hours of the attack, often reaching a peak level in 24 hours. In most instances of pulmonary infarction the transaminase levels are not appreciably increased. When they are, the level is relatively low and the peak concentration occurs between the 3rd and 6th days, in contrast to the much earlier rise and higher values in acute myocardial infarction.

The electrocardiogram in pulmonary embolism may simulate that of posterior wall infarction but is easily distinguished from the latter by the presence of a deep S_1, inverted T wave in leads V_1 to V_3, a shift to the left of the transitional QRS in the chest leads, a late R in aVr, and the absence of a Q in aVf. Shortly after the attack subsides these changes rapidly revert to normal or the previous pattern.

Next to cough, chest pain is the second most common complaint in patients with tumors of the lungs and mediastinum. Mayer, Rappaport, and La Due (21) found that 21% of 824 patients gave a history of chest pain as a presenting complaint and that almost all patients developed the symptom sometime in the course of the disease. These authors make the important observation that vague chest discomfort, without actual pain, is an early symptom in even a higher percentage of cases before roentgen changes are found.

The pain is described as a fullness, an aching, or a pressure sensation, at first transient, later more persistent. It is often more apparent after exercise and it is sometimes associated with changes in position, cough and deep breathing, in this respect simulating root chest pain.

Chest pain of this type, when associated with cough, intermittent wheezing and mild general symptoms such as aching muscles and joints, fatigue, low-grade fever and recurring respiratory infections, should raise a suspicion of lung cancer. At this stage physical signs may be absent or minimal. Partial bronchial obstruction may result in diminished breath sounds, localized wheezing, inconstant rhonchi and rales. Roentgen studies of the chest may be negative or show only questionable deviations from the normal such as transient opacities, enlargement or irregularity of the hilar shadows, hazy infiltrations along the vascular trunks or linear atelectasis. Such findings demand watching and further roentgen studies. Cytologic studies of bronchial washings and sputum, bronchoscopy or exploratory thoracotomy may be indicated.

In the more advanced stage when the primary tumor or metastasis has invaded or compressed adjacent bronchi, lungs, pleura, chest wall or other structures of the mediastinum, a variety of symptoms and signs may appear. Tumors may exert pressure against the lower cervical and upper thoracic sympathetic trunks (Horner's syndrome), recurrent laryngeal and phrenic nerves, esophagus, superior or inferior vena cava or the heart. They often cause constant substernal pain. When the upper mediastinum is involved, pain is felt at the base of the neck or at the upper anterior chest. It may be accompanied by cough, hoarseness, dyspnea, dysphagia, neck and upper chest cyanosis, venous distention and edema. At times substernal

crepitation or friction sounds are noted on auscultation. A syphilitic saccular aneurysm of the transverse arch of the aorta also produces the same upper mediastinal symptoms but is readily differentiated from a tumor by angiography.

Carcinoma of the lungs may also be associated with clubbing of the fingers and, when there is metastasis, nodes may be found in the supraclavicular region, neck and axillas. Superior sulcus tumors (Pancoast tumors) commonly involve the sympathetic trunks producing a Horner's syndrome, and infiltrate the ribs, vertebrae and brachial plexus causing severe and constant pain in the shoulder and upper extremity that is soon followed by weakness, paresis and atrophy. The distribution of pain at first suggests cervical root compression or a deltoid bursitis. Tenderness over the bursa and over lower cervical and upper thoracic spine, however, are absent, and special maneuvers of the neck do not induce symptoms unless the vertebrae have been invaded.

Since chest pain may be an early presenting symptom in carcinoma of the lungs and since early diagnosis is of paramount importance, the possibility of this increasingly common disease must be constantly kept in mind. Chest pain that cannot be definitely attributed to other conditions must be observed in relation to such symptoms as cough, wheezing, malaise and recurring respiratory infections. Repeated chest plates, cytology studies and bronchoscopy may be indicated.

MEDIASTINAL EMPHYSEMA

The most common symptom in mediastinal emphysema is chest pain, which must be differentiated from that of angina pectoris, acute coronary failure, myocardial infarction, dissecting aneurysm of the aorta, pericarditis, mediastinitis and pulmonary embolism. According to studies by Macklin and Macklin (22), this condition occurs when overinflated marginal alveoli contiguous with bronchi, bronchioles, blood vessels or pleura rupture into the underlying connective tissue. From here air is transported by way of the sheaths of the pulmonary blood vessels (interstitial pulmonary emphysema), and not the sheaths of the bronchi or bronchial blood vessels, to the mediastinum. As the mediastinal pressure increases above the atmospheric pressure it ruptures into (1) the pleural space, producing a pneumothorax usually left-sided or (2) the subcutaneous

tissue of the head, neck, chest, abdomen, back or extremities.

Aisner and Franco (23) list the following precursors or conditions that result in rupture of the marginal alveoli. (1) Atelectasis with compensatory hyperinflation of adjacent alveoli occurring in infectious diseases, inhalation of foreign bodies, neoplasms of the lungs, atelectasis of the newborn and bronchial asthma. (2) Increased alveolar pressure resulting from forceful respiratory efforts against resistance, as in closure of the glottis during acts of straining, coughing or violent exercise, laryngospasm, acute obstructive laryngitis, bronchial asthma, playing of wind instruments or glass blowing. (3) Tracheal insufflation as in intratrachial anesthesia, resuscitation of newborn, drownings, etc., by methods of intratracheal insufflation and use of the pulmotor, and positive pressure inhalation therapy. (4) A combination of these conditions. The presence of any of these circumstances preceding an attack of severe anterior chest pain bears consideration in differential diagnosis.

Pain may be mild or severe. It may be sharp, stabbing and pleuritic or oppressive and squeezing. Onset is usually sudden but it may be gradual. The commonest locations are the precordium, sternum, the axillary and midscapular areas. Pain may radiate to the neck, left shoulder and upper extremities or to the back. It is influenced by body positions, intensified by lying on the left side or relieved in the upright position. It may vary in duration from minutes to hours. When the air escapes into the subcutaneous tissue, mediastinal pressure is released and the pain gradually subsides.

In addition to pain, dyspnea is a striking symptom. It depends on the degree of interstitial emphysema and splinting action of air in the connective tissue of the lung. The circulation of blood may also be hampered by compression of the vena cava and pulmonary veins and direct compression may produce a tamponade effect impairing coronary flow and producing the pain of coronary failure.

The physical examination may reveal crepitation over the mediastinum synchronous with the heart beat or the respiratory movements, obliteration or decrease in cardiac dullness, left-sided pneumothorax or subcutaneous emphysema. Air in the mediastinal tissue produces peculiar and unusual sounds that have been described as crackling, clicking, bubbling, crunching, crinkling and snapping. The sounds are most readily detected

in the lateral recumbent position and during the expiratory phase of respiration. The diminished cardiac dullness or hyperresonance over the heart is best elicited in the recumbent position. The presence of subcutaneous emphysema should always suggest the existence of mediastinal emphysema. Subcutaneous emphysema usually involves the neck, supraclavicular and infraclavicular fossas and axillas, but it may be much more extensive. One case reported by Aisner and Franco involved the head, neck, chest, abdomen, back, lower extremities and genitalia. The temperature is usually normal but occasionally minor elevations occur.

The blood pressure is usually not altered unless the increased mediastinal pressure produces a tamponade effect, in which case it may fall. The electrocardiograms show nonspecific changes in approximately one fourth of the cases with deviations in the ST segments and T waves, diminished voltage of the R wave in the left chest leads and shifts in electrical axis. Some changes are related to the mediastinal shift that accompanies the pneumothorax. These electrocardiographic changes return to normal as the air is released or absorbed in the course of days or weeks.

A single roentgen examination of the chest in this condition may be negative. When the diagnosis is suspected serial roentgenograms must be taken and lateral views of the chest are necessary to demonstate air trapped behind or in front of the heart. Air is often visible as streaks of increased radiance running along the borders of the heart. Subcutaneous emphysema is readily demonstrated by air pockets or streaks in the fascial planes of the subcutaneous tissue. The roentgen examination may demonstrate a partial pneumothorax not clinically apparent.

SPONTANEOUS PNEUMOTHORAX

Spontaneous pneumothorax occurs at rest or after exertion, a paroxysm of coughing, laughing or straining. Onset is sudden with respiratory distress and pain on either side of the chest. The pain is usually sharp and cutting but it may also be felt as a substernal pressure, an expanding discomfort or a sense of intrathoracic constriction, particularly when a tension pneumothorax has developed. The diagnosis is quickly established on the basis of physical signs and roentgen findings. Resonance

is increased and respiratory sounds are diminished or absent over the involved side. The apex impulse, trachea and mediastinum are displaced toward the opposite side.

MASSIVE COLLAPSE OF THE LUNGS

Atelectasis of the lungs occurs in varying degrees in association with several pathologic conditions of the lung, but collapse of whole lobes or the entire lung occurs only in the presence of paralysis of the diaphragm, foreign bodies in the bronchi, or following certain injuries or surgical operations. Onset is sudden, occurring a few hours to several days after injury or operation. Symptoms may be few or even absent. In some patients, however, the attack is characterized by pain and tightness over the lower anterior, lateral or posterior chest wall, dyspnea, cough and cyanosis. The physical examination reveals dullness to flatness at the base of the involved lung, displacement of cardiac dullness and apex impulse to the involved side, and a comparative immobility of the affected side during respiration with retraction of the interspace. Over the opposite side there are hyperresonance and exaggerated breath sounds. Over the involved side breath sounds may be diminished or they may be bronchial in character. Rales are usually absent at first but later, with the onset of a productive cough, fine and coarse rales appear. Minor rises in temperature, respiration and pulse may occur soon after the onset. The diagnosis is confirmed by the roentgen findings of diminished radiance over the affected area, marked displacement of the heart and trachea in the same direction and an upward merging of the diaphragm with the involved area. As the chest clears, the diaphragm is observed to be elevated and its excursions are diminished or absent.

ACUTE FIBRINOUS PLEURISY

An acute fibrinous pleuritis may occur as a primary inflammation of the pleural membranes or be secondary to a local disease process in the lungs or mediastinum or to some systemic or chronic illness such as leukemia or nephritis. In every case the possible presence of a primary process elsewhere should be investigated.

An acute pleuritis is characterized by the sudden onset of

sharp, cutting, stitch-like chest pain that is aggravated by inspiration or any act or bodily movement that increases respiration. Pain is more commonly located over the lower lateral aspect of the chest wall from the fourth to seventh interspaces. When localized in the upper chest or apical region pain may be referred to the shoulder. When the diaphragm is involved pain may be referred to the abdomen or neck. The immediate response on the part of the patient is rapid shallow respirations to reduce discomfort. Pain is also regularly relieved by any external splinting device such as a tight binder that reduces chest movements.

The pleuritis is most often accompanied by cough and malaise, chills or fever may be present. The cough is most often dry and hacking. Fever is usually of low grade or it may be absent. Physical examination usually reveals a friction rub that is synchronous with respiration. The most common site of the rub is the fifth and sixth interspaces in the axillary line. The leukocyte count may be normal, borderline or elevated, at times rising as high as 20,000. The diagnosis is readily made on the basis of the sudden onset of sharp pain aggravated by respiration and the presence of the friction rub. Diaphragmatic pleurisy is more difficult to diagnose, for often a friction rub is not heard. On fluoroscopic examination, however, the involved area of the diaphragm may be seen to be immobile and elevated.

The pain of acute pleuritis must be differentiated from that due to root compression and herpes zoster. Root compression may produce the same sharp pain along the lateral aspects of the chest wall, aggravated by inspiration or body movement. A friction rub is absent and the objective signs of root compression and the response to traction establish the diagnosis. Herpes zoster, with involvement of the upper thoracic ganglia and roots, may also produce sharp or dull pain over the lateral aspects of the chest wall. More often the pain is not aggravated by respiratory movements. It commonly precedes the onset of the herpetic eruption by a day or two and diagnosis is usually doubtful until the characteristic eruption with its dermatome distribution appears.

Occasionally an acute pleuritis with diaphragmatic involvement must be differentiated from acute inflammatory conditions in the abdomen. The close relationship of the pain to respiration, its relief by splinting of the chest with a binder

and the indefinite character of abdominal findings usually point to the correct diagnosis. An acute pleuritis also occurs as an acute contagious infection that may reach epidemic proportions. Epidemic pleurodynia is characterized by the sudden onset of fever, headache and pain at the level of the attachment of the diaphragm, over one or both sides of the lower anterior chest and high in the epigastrium. The pain here is likewise aggravated by inspiration, cough and body movements and relieved by splinting the lower thorax. The fever may rise to 104 degrees within a few hours and drop to normal within 24-48 hours. Recurrences are common and intermittent fever may continue for several days. Epidemic pleurodynia also is sometimes confused with acute abdominal emergencies and, when nausea, vomiting and distention are added to the picture, differential diagnosis may be difficult. The epigastric tenderness in pleurodynia is superficial in contrast to the deep tenderness that is present when the abdominal viscera are the site of symptoms and the decided relief of pain by splinting the chest with a binder usually points to the correct diagnosis.

ROOT CHEST PAIN

Once one becomes alerted to the syndrome, root compression will be found to be one of the common causes of chest pain. Illustrating its incidence when a cardiologist is well aware of it is Ollie's (24) report of 197 cases in a survey of 600 consecutive patients complaining of chest pain. The fact that it so often simulates various manifestations of coronary disease makes it particularly important in differential diagnosis.

In the vast majority of cases, root compression results from faulty body mechanics, postural or occupational strain and trauma, which, in time, cause the soft tissue and bony changes of osteoarthritis of the spine. Degenerative disk disease with or without protrusions, spurs about the posterior vertebral margins and the interarticular joints, deformities of the root pouch or fibrosis of the root sleeve are conditions that most commonly produce root irritation with chest pain. Primary neoplasm of the spinal cord and meninges and metastatic lesions from such primary sites as the prostate, lungs or gastrointestinal tract may also be responsible for root compression and these sources must be considered in diagnosis.

A rough idea of the possible role of some of these factors

can be obtained from plain roentgenography and sometimes more exact information from myelography. The age, occupation, presence or absence of trauma, type of onset, duration and severity of symptoms and signs and their response to therapy give some information as to the probable character of the lesion.

Frykholm's (25) studies show that compression of the ventral motor roots produces pain in the distribution of the muscles supplied by these roots and this type of pain differs in character from that due to dorsal sensory root compression. Motor root pain is usually dull, gnawing, aching, continuous and more localized. Sensory root pain is intermittent, sharp, stabbing and frequently radiates down the upper extremities. Another form of pain, more superficial in character, most often described as a tingling or "pins and needles" sensation, and frequently associated with numbness of the hand and fingers, is also of sensory root origin. Motor and sensory root pain may co-exist.

Compression of lower cervical motor roots produces pain in the region of the serratus and pectoral muscles that are innervated by these roots. The skin of the chest wall and the intercostal muscles are supplied by the upper thoracic sensory roots. Therefore, chest pain may arise from either the lower cervical or the upper thoracic regions of the spine. The higher incidence of roentgen changes in the lower cervical spine in contrast to the upper thoracic spine suggests that the lower cervical roots are more often the cause of symptoms. The frequent occurrence of chest pain with motor root characteristics also suggests a lower cervical origin.

GENERAL CHARACTERISTICS OF ROOT PAIN.—Chest pain due to root irritation may be mild or severe, sharp or dull, intermittent or prolonged, localized or widespread and occur with or without radiation. When sharp, it is usually described as a pinching, piercing or stabbing; when dull, as a soreness, pressure or tightness, or a constricting or expanding discomfort. The sharp form of pain often radiates; the dull form more often remains localized. When mild, especially when the patient is more concerned with the significance than the discomfort, it is sometimes erroneously attributed to an anxiety neurosis. When severe it may simulate myocardial infarction, coronary failure, angina pectoris or pleurisy (26, 27, 28). More often it is the prolonged dull, constricting distress that arises from irritation of the ventral motor roots that suggests coronary disease. When, in addition, the sharp radiating pain of dorsal

sensory root compression is superimposed, as it often is, the simulation of coronary disease is even more complete.

Pain of spinal origin may occur over any part of the chest, in the back on either side of the spine, in the axilla, in front over the breasts and substernally. It is most often bilateral and more commonly felt in the anterior chest wall. It may first appear in the back or along the axillary lines and radiate around the chest wall to the sternum or, less commonly, straight through the chest to the sternum. Occasionally pain is felt simultaneously in front and in back and sometimes in back after first appearing in front. It is frequently localized to the substernal region, the precordium or the corresponding area of the right chest. It may radiate from sternum to axilla, shoulder girdle, inner or outer aspect of arm, forearm and fingers or start anywhere in the extremity and radiate up to the anterior chest wall. Occasionally it radiates from sternum to jaws or neck region, where it may be felt as a choking sensation. Severe attacks may be accompanied by pallor and excessive perspiring. In some patients the chest pain is precipitated by walking, simulating angina pectoris. Many patients also complain of a peculiar respiratory distress characterized by an inability to breathe in or out (29). It may be associated with other cervical root symptoms such as shoulder girdle pain, occipital headache or vertigo (30).

CARDINAL CHARACTERISTICS AND DIAGNOSIS.—In addition to the many features that suggest coronary disease, certain cardinal characteristics of radicular pain are usually present. Attacks frequently occur: (1) in relation to certain movements or postures, such as bending, turning or prolonged sitting in a crouching position, (2) after coughing, sneezing, deep breathing or straining at stool and (3) after hours of recumbency, often waking the patient from sleep. These relationships are not always volunteered. The patient has to be questioned to bring out this information and sometimes significant data are obtained only when subsequent attacks are observed.

Even when some of the cardinal characteristics are present, and they are not present in every case, the diagnosis is always based on additional data. The most important is the reproduction of attacks by application of pressure over the lower cervical or upper thoracic vertebrae. Such attacks can be reproduced in many patients with recently acute symptoms and the induced pain often has the location, distribution and character of

the spontaneous attacks. Tenderness over the involved segment of the spine, and over the anterior chest wall in the region of the parasternal line or axilla, is regularly present. Finally, the diagnosis can be confirmed by a striking response to therapy, consisting of traction of the cervicothoracic spine, postural correction and exercises.

In discussing the specific manifestations of the syndrome as it simulates other causes of chest pain, case studies will perhaps best illustrate the problems one meets in differential diagnosis.

ROOT PAIN SIMULATING MYOCARDIAL INFARCTION.—The root syndrome may appear suddenly as an overwhelming attack of severe chest pain without any previous history to suggest a disorder of the spine. When such severe attacks are isolated or infrequent, the cardinal characteristics of the syndrome may not be apparent. The following case illustrates such an acute process with minor attacks culminating in a major episode of such severity as to suggest myocardial infarction. In this patient, even during the acute episode, questioning revealed that there was a significant relationship between body positions or movements and intensity of pain.

Case 1.—A 51-year-old male executive was seen in consultation for what appeared to be an attack of acute myocardial infarction. Earlier that day, while sitting in his office, he developed severe chest pain located substernally and just to the left of the sternum, where it was most intense. It was viselike in character and did not radiate. He felt as though "a weight was compressing his chest." The pain persisted for 2½ hours before he obtained appreciable relief. His associate noted he was "white as a sheet" and that he perspired profusely. He was helped home, seen by his physician and given morphine. Later, he recalled that while resting in a chair during the attack, he could relieve the pain somewhat by bending a little forward. After getting into bed, he noted that lying on his left side regularly increased the severity of the pain.

Three weeks earlier, he had begun to have attacks of pain of a similar character, but less severe and of shorter duration. The pain had started in the lower axillary region on both sides, squeezed him as in a vise and then localized in the substernal area. He had had approximately 12 attacks of this kind, lasting from 30 to 90 minutes, and in the past few days they had become more frequent and severe. They were not precipitated by walking but, once started, definitely aggravated by exertion. Two or three attacks awakened him from sleep, but most of them occurred during the day, most often while he was in a sitting position. The past history was irrelevant.

When seen several hours after the onset of his recent major attack,

he still complained of mild substernal distress. Physical examination of the chest was not remarkable. Blood pressure was 170/110. Examination of the spine showed a moderate dorsal kyphosis. Pressure over the seventh cervical and first and second thoracic vertebrae revealed marked local tenderness and induced agonizing substernal pain of the character and distribution noted in the attack described. He was angry and annoyed by the procedure. Even light pressure produced viselike pain and substernal distress. An electrocardiogram taken later was within normal limits. X-rays of the cervical and thoracic spine showed the changes of advanced osteoarthritis. Orthopedic treatment was advised and, except for 3 mild attacks in the next 6 months, there was no recurrence of severe symptoms in the next 9 years that he was followed.

ROOT PAIN SIMULATING ATTACKS OF ACUTE CORONARY FAILURE.—When root irritation is chronic, a patient may have innumerable bouts of chest pain lasting seconds, minutes or hours. With this number of attacks the cardinal features of radiculitis, namely, pain in relation to recumbency, positions and movements, is more likely to be apparent and this information should suggest root disease. Early in its course, however, when attacks are isolated and few in number, these relationships may not be apparent and the attacks will closely simulate acute coronary failure.

Case 2.—A 54-year-old housewife complained of repeated attacks of severe substernal and anterior left chest pain, of 3 years' duration, which had been attributed to coronary disease. They occurred most often at night, forcing her to sit up or walk about. The pain usually started in the mid-dorsal region of the chest posteriorly, moved around the left breast and up under the sternum, causing substernal pressure and choking or the sensation of a lump in the neck. Less frequently it started in the substernal region and radiated to both jaws, where it was felt as numbness as well as pain. She often exclaimed, "My face feels paralyzed!" At times there was radiation down the left arm to the elbow. During attacks there was marked hyperesthesia over the precordium and she often held up her left breast to avoid its pressure against the chest wall. Coughing or sneezing caused a burning pain across the base of her neck. Attacks often lasted an hour, occasionally several hours. They were less frequent during the day and usually occurred when in a sitting or bent-over position. Walking about or straightening up regularly gave relief. On 3 or 4 occasions in the past 2 years, she had attacks in the course of walking fast or climbing stairs; these were relieved by stopping.

Physical examination of the heart and lungs was not remarkable. Blood pressure was 180/100. An electrocardiogram was within nor-

mal limits. A 7 ft. roentgenogram showed slight enlargement of the left ventricle. Examination of the spine showed a markedly increased dorsal curve. Motion of the neck was restricted in all directions. Coughing caused pain at the base of the neck in back. Slight pressure over the second and fourth thoracic spinous processes produced severe anterior chest pain similar to that experienced in her spontaneous attacks. It was immediately felt in the midsternal region, under the left breast, in the throat region and along the left jaw as high as the temporomandibular joint. Substernally it felt like a constriction. Slight pounding over the second or third thoracic vertebrae produced sharp pain, which stabbed from the back straight through to the sternum and from there up to the neck, left jaw and frontoparietal aspect of the head.

Following a course of overhead traction, postural correction and exercises there was striking improvement and in the course of the next year only occasional recurrences of mild pain.

ROOT PAIN ASSOCIATED WITH RESPIRATORY DISTRESS.—Respiratory distress characterized by the inability to inspire or expire is a common manifestation of lower cervical and upper thoracic root compression. Approximately one third of patients with chest pain of root origin complain of this type of distress at some time in the course of the illness. It occurs not only with the attacks of severe pain but sometimes without pain (29). Rarely it is a presenting symptom long before the onset of chest pain and attacks may be sufficiently severe and persistent to suggest erroneously cardiac asthma.

The patient is usually aware that he is unable to inspire or expire and that he must force himself to breathe, "My breath is cut off," "I am unable to take a deep breath," "my chest seems fixed," and "I find I must force myself to breathe" are common expressions. This type of respiratory distress can frequently be induced with or without chest pain by the application of pressure over the lower cervical or upper thoracic spine.

Patients with coronary disease also sometimes complain of respiratory distress. The combination of this symptom and chest pain therefore tends even more strongly to suggest coronary disease and many patients with this combination of symptoms have thus been erroneously treated.

Case 3.—A 50-year-old housewife gave a history of "shortness of breath" and attacks of severe anterior chest pain of 2 years' duration. She had been under observation for marked hypertension for several years and, 5 years earlier, had had an attack of prolonged left chest pain that was attributed to coronary disease. Her attacks during the past 2 years were ascribed to the same condition.

Most attacks occurred at night and were often associated with respiratory distress. The pain was described as a dull ache. It usually started posteriorly along the inner aspect of the left scapula and radiated anteriorly to the neck and around the left breast, localizing over a small area within the left nipple line. The attacks usually lasted 10 or 15 minutes. Soon after the onset she was "unable to breathe" and could not take a deep breath. At times she gasped in an effort to obtain more air. At the start of each attack, she sat straight up in bed, arched her shoulders back, and at times exercised her arms to obtain relief. She had had a few attacks in the course of walking and climbing hills and sometimes mild substernal pain on getting out of bed in the morning. She was also troubled by posterior neck pain and suboccipital headaches.

Percussion showed the heart to be slightly enlarged. The first sound was accentuated and there was a slight systolic murmur at the apex, a moderately loud systolic murmur at the base and an early aortic diastolic murmur along the left border of the sternum. Blood pressure was 240/120. The lung bases were clear. Neck rotation was appreciably limited to the left. There was slight scoliosis of the thoracic spine to the left with slight angulation at the level of the fourth thoracic vertebra. To the left of this region there was muscle spasm and pressure at this point of angulation produced wincing pain anteriorly in the region of the third left rib and parasternal line. A similar but less severe pain was produced by light pounding with the fist over the second to seventh thoracic vertebrae. With the onset of pain in each instance, respiration became rapid, shallow and gasping.

An electrocardiogram showed no deviation from the normal. Roentgenograms of the cervical and thoracic spine showed moderately advanced osteophytes along the anterior and posterior margin of the cervical vertebrae, narrowing of the fourth to seventh disks, osteoarthritic changes in the intervertebral joints and scoliosis. In the thoracic region there were anterior margin osteophytes, narrowing of the fifth to eighth disks, scoliosis and kyphosis.

She was later seen by an orthopedist who noted a slight limp, a pelvic tilt with the right iliac crest higher than the left, scoliosis in the lumbar area to the right which had caused the compensatory scoliosis in the thoracic spine to the left, producing slight angulation at the level of the fourth vertebra. The left lower extremity was ¾ inch shorter than the right. Orthopedic treatment, consisting of heel lift on the left foot, bed boards, cervical traction and manipulation and moist heat gave immediate partial relief. After a few weeks of treatment, pain was almost completely abolished. Seen 7 months later she was active, with only mild discomfort on rare occasions.

ROOT PAIN SIMULATING CORONARY FAILURE WITH "ATYPICAL ANGINA PECTORIS."—When patients have attacks of chest pain

at rest and in addition chest pain on walking or on exertion, coronary failure with angina pectoris is suggested and the diagnosis of coronary disease appears all the more complete. The one set of symptoms appears to lend corroboration to the other and, although the history shows that the pain in walking does not exactly conform to all recognized criteria for true angina pectoris, the tendency is to label it "atypical angina pectoris." While we know that atypical angina pectoris may sometimes indicate coronary disease, we also are familiar with the many times it indicates doubt in the physician's mind and presents problems in differential diagnosis with such conditions as diaphragmatic hernia, gallbladder disease, psychoneurosis or the radicular syndrome.

Some patients with root compression maintain that the pain in the course of walking is relieved by stopping, and this further suggests angina pectoris. On careful inquiry, however, it is usually disclosed that pain is not always immediately relieved by stopping and, when it appears to be, stopping is accompanied by a "straightening-up" with hyperextension of the spine. The pain is usually longer in duration than in angina pectoris. More important is the fact that it is not closely related to cardiac effort, for the same patient is capable of more severe tests of physical exertion without precipitating chest pain. Unlike the usual onset in angina pectoris, chest pain usually occurs first at rest or in bed or is associated with bending or stooping long before it occurs in the course of walking.

Case 4.—A 51-year-old retail store owner was first seen in 1943 for an evaluation of his cardiac condition. Five years earlier, while walking in a snowstorm, an attack of severe substernal pain had forced him to stop and was apparently relieved by resting. Ten minutes later a second attack forced him to stop, this time the pain radiating to the left arm. Shortly after getting into bed that evening, he had severe "crampy" pain that persisted for 15 minutes and, with it, respiratory difficulty described as "an inability to breathe." An able cardiologist at that time had made a diagnosis of coronary disease. The electrocardiogram had been normal.

Further history revealed that for several years before the diagnosis and in the 5 years since, he had occasionally had similar substernal distress, although not as severe, on stooping or bending, in the course of walking, or after prolonged sitting as in playing cards. He described the pain always as "crampy," occasionally radiating to the left arm.

Physical examination and an electrocardiogram were normal. The

spine was not tender and areas of chest wall tenderness were not looked for at this time (1943). Blood pressure was 130/80.

The following winter he had 2 attacks during long walks following large meals. These were relieved by stopping. Questioning brought out the fact that when he stopped he "straightened up," throwing his shoulders back. He attributed the relief to this act and maintained that the earlier attacks before the diagnosis of coronary disease had also been relieved in this way. During the next few years electrocardiograms before and after exercise were negative. In 5 separate studies he went from 30 to 40 trips on the two-step staircase, an ice cube in his left hand, without having chest pain or showing electrocardiographic abnormalities within 10 minutes after exercise.

Several times during this period he also complained of bouts of vertigo, shoulder pain and low back pain. In 1947, he awoke one night with sharp pains over the left precordium and axilla that were aggravated by certain positions or movements in bed. The next day the same pain occurred with yawning, deep breathing and straining at stool. At this time the thoracic spine from D2 to D8 was markedly tender, but no pain was referred to the anterior chest wall. There was tenderness on both sides over the region of the costochondral junction of the ribs. Roentgen studies showed advanced hypertrophic changes with extensive spur formation about the margins of the cervical and thoracic vertebrae. The middle and lower cervical vertebrae were roughened and the intervertebral spaces in these regions were narrowed and irregular in outline.

Therapy for root compression, consisting of traction, postural correction and exercises, was effective in abolishing the spinal tenderness and, for the most part, the attacks of substernal pain for 7 years.

In 1954, the patient again had repeated attacks of prolonged substernal pain but, unlike the attacks in the past, these were not influenced by straightening up or changing position. Examination of the spine did not show appreciable tenderness and there was only slight parasternal tenderness over the first and second left ribs. Electrocardiograms were negative. Test traction during 2 bouts of pain brought no relief. He was hospitalized, and during the next few days serial electrocardiograms showed a posterolateral myocardial infarct.

ROOT PAIN SIMULATING ANGINA PECTORIS.—There are instances, fortunately few in number, in which a thoracic root syndrome may so closely simulate typical angina pectoris that differential diagnosis is extremely difficult. Early in its course, the chest pain may occur only in walking or other exertion and be relieved quickly by rest. The type of pain may be identical in location, character and radiation with that of angina pectoris

and its occasional response to nitroglycerin may be further misleading. Although it is seldom precipitated by excitement or cold, association with even these factors is occasionally made by patients.

When the pain is induced by walking or other exertion, it must be re-emphasized that the relation is not to cardiac work performed but to certain spinal movements that increase root irritation. Each step in walking apparently causes a little jar to the spine, particularly when the patient assumes an exaggerated kyphotic posture. The absence of attacks in the same patient in the course of greater physical exertion points to a spinal factor rather than to angina pectoris.

The time factor in the relief by rest or in the occasional response to nitroglycerin is very important. In angina pectoris relief is most often immediate; in root pain it is rarely ever as quick. The associations made by patients with excitement or cold can often be traced to certain body movements or postures at the time; or they are coincidental.

Case 5.—A 25-year-old housewife was seen in 1946 complaining of attacks of substernal pressure discomfort of 2 months' duration. The attacks were precipitated by walking or climbing stairs and were completely relieved by rest within 30 to 60 seconds after stopping. They occurred particularly in cold weather. There was a past history of joint pain at the age of 7, when a heart murmur was detected and a question of rheumatic fever was raised.

Physical examination showed a tall, thin woman with an exaggerated kyphotic posture. The heart was not enlarged. At the apex there was a grade II systolic murmur. There were no diastolic murmurs before or after exercise. Examination of the spine showed dorsal kyphosis with flattening from D1 to D7. Over D6 and D7 there was appreciable tenderness and tenderness was also present anteriorly in the region of the third and fourth costochondral junctions on both sides of the sternum.

The resting electrocardiogram was within normal limits. On the two-step staircase she developed mild substernal distress after the seventh trip which continued without increasing in severity up to 17 trips and for 2 minutes after the test.

During the next few weeks she had occasional attacks while walking but only when she felt particularly cold. There were no attacks at rest or in bed. Six months later, however, she complained of upper and midsternal pressure of 10 days' duration. This pain was of the same character but now it occurred not only in walking but intermittently throughout the day at rest as well as with activity. It was definitely aggravated by bending and provoked by coughing. Physical examination of the heart was unchanged. On getting up

from a sitting position, in the course of the interview, she developed mild substernal distress which was quickly relieved by hyperextending her spine. A diagnosis of root pain was made. She was referred to an orthopedic surgeon who gave her a course of traction. This immediately stopped the pain she had with bending but not the attacks in the course of walking.

She was asked to keep a close record of these attacks to determine their relationship to exertion, excitement and cold. She reported that the relation to walking in cold weather seemed striking. Straightening up and throwing her shoulders back appeared to relieve the pain but did not abolish it. On one occasion this act seemed to spread the pain over a wider area, particularly when she continued to walk. Relief was always immediate when she stopped. Nitroglycerin taken shortly before walking did not prevent the attacks.

Shortly after this, she reported that she had bought a warm coat and, when wearing it, was able to walk longer distances in the cold without pain. She stated that previously she had always brought her shoulders downward and forward, tensing her chest muscles in response to cold weather and suggested that perhaps she had obtained only partial relief from throwing her shoulders backward because she never relaxed her muscles completely. Once she tried walking on a cold day with her coat unbuttoned and developed a typical attack. She was conscious of having drawn her shoulders down and forward in response to the cold. With the warm coat there were no attacks in contrast to the winter before, when she might have had several in the course of a 15 minute walk.

She has now been under observation for over 11 years. During this time she has been free from pain for periods of 1 to 3 years.

ROOT PAIN SIMULATING ACUTE PLEURITIS.—When root pain is referred to the lower lateral aspect of the chest and brought on by or aggravated with each inspiration, acute pleuritis is simulated. Friction rubs are not heard and examination of the lungs is negative but the possibility of a viral pleuritis is not thereby excluded. The absence of a friction rub is sometimes erroneously attributed to diaphragmatic involvement. The following patient had root irritation with chest pain that appeared primarily in relation to respiratory movements. The response to painful inspiration was rapid shallow breathing such as one commonly sees in acute pleuritis.

Case 6.—A 50-year-old woman was seen postoperatively complaining of chest pain of 24 hours' duration. Two days earlier operation for a ruptured ovarian cyst had been performed. The following day she had suddenly developed moderately severe pain in the lower right axillary region. At times this pain extended under

the right breast and also posteriorly just beyond the posterior axillary line. It was somewhat sharp and regularly intensified by inspiration. When she held her breath it abated and at times it was necessary to do so to obtain relief. For this reason she carefully avoided deep breathing. The night before she had been unable to sleep in a reclining position but experienced striking relief when propped up. Since the onset of the chest pain she had had constant nausea.

Her physician suspected an acute pleuritis, nonspecific, or part of a pneumonic process, but there was no rise in temperature or change in blood count. When examined, the patient was anxious and uncomfortable. Breathing was shallow and respirations increased to 24 per minute. Examination of the heart was not remarkable except for a grade II systolic murmur at the apex. The lungs were clear and a roentgenogram taken later was negative. There was marked tenderness over the right and left fourth costochondral junctions anteriorly and over the fourth thoracic vertebra posteriorly. Pressure over this region of the spine on 3 different occasions reproduced pain under the left breast and intensified the right axillary pain on inspiration.

Following traction in a semi-reclining position, pain on inspiration was immediately relieved and disappeared entirely in the next few hours.

CHEST PAIN AND PAROXYSMAL COUGH OF ROOT ORIGIN.—Rarely cough is associated with spinal root chest pain. The following patient strikingly suggested his own diagnosis by suspending himself from an overhead rail, thus giving himself the benefit of traction.

Case 7.—A 39-year-old meat handler complained of chest pain with exertion of several months' duration and paroxysmal cough of 3 weeks' duration. At first, pain was confined to the anterior right chest region and occurred regularly in the course of his work. Later, he developed a gripping pain to the left of the upper sternum in the course of walking. On several occasions it forced him to stop and this gave immediate relief. The coughing spells of the past 3 weeks were accompanied by right anterior chest pain. Attacks now also occurred in the morning after getting out of bed. In the course of his work, involving considerable exertion, he found that raising his arms gave a little relief and, later, that swinging from an overhead meat rail gave total relief and stopped both paroxysms of cough and chest pain. His discovery was so striking that he adopted the same procedure to relieve his morning spells at home.

Physical examination showed a tall, heavy-set, muscular man, slightly overweight. The heart and lungs were normal. There was marked parasternal tenderness over either side of the sternum in

the region of the second to fourth costochondral junctions. There was moderate tenderness over the second thoracic vertebra.

During examination he complained of mild right chest pain aggravated by deep breathing. On the exercise tolerance test he went 40 trips without developing pain and electrocardiograms were negative before and 10 minutes after exercise. During overhead traction, upper right chest tenderness was entirely relieved and after traction he maintained he could take a deep breath without pain.

He was given overhead traction daily, a regimen of exercises, and changed from three pillows to one for sleeping. When rechecked 5 days later, chest pain and cough had almost disappeared and parasternal tenderness was no longer elicited. There was little or no tenderness over the thoracic spine. When asked to cough he could do so without pain. During the past 4 years neither cough nor chest pain have recurred.

ROOT CHEST PAIN FOLLOWING TRAUMA.—Root chest pain is often precipitated by trauma and appears after comparatively minor injuries to the shoulder girdle and trunk. Attention is first directed to the bruises and soft tissue injury over the shoulder, arms or chest wall and sometimes only hours or days later will chest pain or other symptoms of root compression first appear. If the spine is not carefully examined these symptoms will be erroneously attributed to muscle injury or other conditions such as heart disease or pleurisy.

DIAGNOSTIC SIGNS OF ROOT COMPRESSION

Several objective findings help to establish the presence of a nerve root disorder: postural abnormalities, tenderness over the spine and in the distribution of pain, the reproduction of pain by certain maneuvers, sensory and motor changes of a segmental character and the therapeutic response to traction. Postural abnormalities and tenderness are not in themselves sufficient to establish the diagnosis, for these signs may exist in latent form without producing symptoms and they frequently coexist with other causes of chest pain.

POSTURAL ALIGNMENT AND FLEXIBILITY.—As postural abnormalities are most often associated with root chest pain they should be looked for. Deviations in the upper spine are almost always associated with abnormal positions of the pelvis and lower extremities. Hence, the postural examination should include the entire body alignment in the standing position. An

anterior pelvic tilt and lumbar lordosis produces a compensatory kyphosis and a forward projection of the head; a lateral pelvic tilt and lumbar scoliosis, a compensatory scoliosis in the upper thoracic and cervical region; and pelvic shifts produce corresponding deviations in the upper spine.

Postural deviations are commonly associated with muscle imbalance and spasm and the range of spinal motion may be impaired. All movements should be carried out and limitations, if present, determined. In lesions of the lower cervical spine, neck rotation is commonly limited and the degree of limitation should be established. Normally the chin can be rotated 90 degrees or more.

EXAMINATION FOR SPINAL TENDERNESS.—Significant tenderness over the spine at the root lesion level is almost an invariable finding when symptoms of root compression are present. In the cervical region the spine should be examined for tenderness over the spinous processes in back and over the transverse processes laterally. The upper cervical spinous processes can be readily palpated in most subjects if the neck is relaxed in a slightly extended position. The transverse processes can easily be felt with the finger tips on each side from just below the mastoid process to the angle of the neck and shoulder. Tenderness in this region is often present when there is none over the spinous processes in back. Examination for tenderness should be made in various degrees of head rotation, flexion and side bending.

Tenderness over the spinous processes can be elicited in the thoracic area with the ball of the thumb. In addition, the thumb is placed in the groove on either side of the spinous processes and pressure directed medially so as to twist the vertebra. This twisting maneuver will sometimes evoke tenderness and referred pain to the shoulder girdle or anterior chest that is not obtained by direct pressure over the spinous process.

In a study by the author of 100 patients with chest pain of root origin, moderate or marked spinal tenderness was present in 94 and the level of tenderness generally corresponded to the distribution of root pain. Since root symptoms only rarely occur without spinal tenderness, in its absence a diagnosis of root disease should be made with caution. Even when the history suggests a root lesion caution in diagnosis is important, for other syndromes may mimic the cardinal symptoms of root disease.

REPRODUCTION OF REFERRED PAIN BY PRESSURE OVER THE SPINAL LESION.—In 33 of 100 patients, pressure directed over the vertebrae induced pain over the anterior or lateral region of the chest wall. Sometimes firm pressure is necessary to elicit this sign and not infrequently it will cause considerable local discomfort. Occasionally, local pain and radicular symptoms will persist for hours, indicating that latent root compression has been aggravated. As this sign firmly establishes the presence of root irritation, however, the procedure is justified.

In many patients the character and distribution of the pain so elicited are the same as the spontaneous symptoms. In other patients the referred pain differs in character and location. This is understandable, for pressure against one vertebra may be transmitted to contiguous vertebrae depending on several local factors. More important, it should be remembered that irritation of a ventral root will induce pain in muscle areas supplied by the root involved. This fact explains the reference of pain over the pectoral region with pressure over the lower cervical vertebrae. It will be recalled that the serratus anterior and pectoralis major and minor muscles are supplied by roots C5 to T1.

INDUCTION OR RELIEF OF SYMPTOMS BY MOVEMENTS.—When a patient states that a specific body movement involving the spine induces chest pain or that pain can be relieved by a given body position, it should be reproduced and observed. Anteflexion of the head on the chest, forward bending of the trunk, reaching movements of the arms and rotation of the head and trunk often induce substernal pain. When active movements do not induce pain, forceful passive movements may do so and should be tried.

In addition to signs that depend on movements of the spinal column, certain special maneuvers are useful. Pain may be produced by the neck compression test of Spurling and Scoville in which the head and neck are tilted toward the affected side and downward pressure is applied over the top of the head. Also, for cervical root lesions, Chavany recommended traction on the extended abducted supinated arm of the affected side with the head tilted to the opposite side. Josey and Murphy found that digital pressure over the major trunks of the brachial plexus induced symptoms. The Déjerine sign, likewise, often but not always suggests a radicular lesion. The patient can be asked to cough, breathe deeply or strain and the effect on symptoms observed.

CHEST WALL TENDERNESS.—Spinal root pain is regularly accompanied by deep skin tenderness. In anterior chest pain of this origin, tenderness is almost invariably present on either side of the sternum, at the parasternal line or costochondral junction. It may also be present in several other areas, particularly the axilla, where it may be most marked. It is often present in the absence of chest pain, then almost always signifying latent root compression, very seldom local disease of the chest wall.

The incidence of anterior chest wall tenderness was recently determined by the author in patients with chest pain of root, coronary and psychogenic origin and in control subjects without chest pain (28). In 100 with root chest pain 98 showed moderate or marked tenderness at one or more costochondral junctions. The effect of overhead traction, tested on 59 of these showed unmistakable reduction or abolition of tenderness in 52 while suspended under traction. In 100 control subjects of comparable age and sex, without chest pain, tenderness was present in only 17. In 100 patients with recent or present pain of coronary origin, tenderness was recorded in 26 and, in 30 patients with psychogenic pain, it was present in 6. Whenever there was opportunity to test tenderness with traction on the control subjects and on the patients with coronary or psychogenic pain, tenderness was relieved as it was in the cases with root compression.

An incidence of 98% in patients with root chest pain suggests its importance in diagnosis. A diagnosis of root compression should seldom be made in its absence. Its presence, of course, does not exclude coronary disease or other causes of chest pain which may coexist with root disease.

After the onset of angina pectoris, coronary insufficiency or myocardial infarction, the presence or absence of areas of chest tenderness should be determined and, when present, the possibility of coexisting root compression considered. With this possibility in mind it will be found that some instances of chest pain in the course of convalescence from coronary disease are not of cardiac origin but can be explained as radicular and treated accordingly.

Deep tenderness over the muscles of the chest wall may be due to several other causes, such as local trauma, muscle strain, inflammation or a malignant process involving the parietal pleura or structures of the chest wall. In the absence of such

lesions, however, chest wall tenderness is almost invariably of root origin. Compression of the ventral roots, as Frykholm has shown, not only produces muscle spasm and pain in the distribution of muscles, but also sets up a local sensitivity or irritability in muscles that is revealed as tenderness. Sometimes pressure over the tender areas produces not only local pain but pain referred to other areas of the chest or extremities (31).

RESPONSE TO TRACTION.—The immediate relief, aggravation or induction of symptoms by traction is an important diagnostic sign. Not only pain, but such root symptoms as vertigo, nausea and respiratory distress, which may accompany root chest pain, are most often dramatically relieved by traction. Physical signs, including muscle spasm and local tenderness, respond in the same way. In a few patients traction will temporarily aggravate or induce root symptoms and this is also of diagnostic value.

In some patients traction will be totally ineffective, especially when compression is due to a hard disk protrusion or a metastatic lesion. A questionable or negative initial response to traction, therefore, does not exclude root irritation as a cause of symptoms.

PITFALLS IN THE DIAGNOSIS OF ROOT CHEST PAIN.—Two possible pitfalls to be avoided in the diagnosis of root compression are: (1) basing a diagnosis on the so-called cardinal signs without sufficient objective confirmation; (2) failure to consider the coexistence of coronary disease. It must not be assumed that chest pain, simply because it is affected by postures and movements, is always of spinal origin. Body movements as well as coughing, laughing, deep breathing and straining may also influence the chest pain in pericarditis, pleurisy, mediastinal emphysema, lung tumors, diaphragmatic hernia, the xiphoid process and manubriosternal syndromes. Regardless of how suggestive the history, the diagnosis should not be made without definite objective confirmation.

When differential diagnosis between coronary disease and root disease is difficult, particularly because the two may coexist or one follow on the other, the physician finds himself in one of the most difficult situations met in practice. His natural wish, of course, is to find the less serious condition. For this reason, even when objective evidence of root compression is demonstrated, it still must be established that this is the sole cause of the chest pain and the coexistence of coronary disease

is not excluded. Furthermore, objective signs of root compression may only indicate latent disease. When doubt exists one should be able to raise questions without arousing false hopes or unwarranted anxiety. The difficulties of the diagnostic problem can be explained to most patients and co-operation obtained. When the diagnosis remains in doubt the effect of traction should be tried. In many cases the complete relief of all symptoms by traction has been the determining factor in excluding the diagnosis of coronary disease.

HERPES ZOSTER

In this acute infectious disease, probably of viral origin, inflammatory changes are found in the dorsal root, root ganglion, and posterior horn of the spinal cord. It is characterized by the sudden onset of pain over the lateral chest wall, followed within a few days by the appearance of a vesicular eruption in the distribution of the pain. Before or with the onset of pain there is often slight fever and malaise, and occasionally mild gastric symptoms. Pain is constant or intermittent, sharp and knife-like and often radiates from the posterior wall around the ribs to the front of the chest. Associated with the pain there may be paresthesias. Examination of the skin soon after onset usually shows hyperesthesia of the involved area. Later this may be followed by decreased sensitivity to touch and pinprick.

The pain may precede the eruption by several days and the diagnosis is usually in doubt until the eruption appears. Both pain and the eruption have the same sensory root dermatome distribution. The fever usually lasts from 3 to 5 days, the eruption from 7 to 10 days. Most often, with the clearing of the eruption the pain disappears. In some cases, however, it may persist for weeks or months. Before the onset of the eruption the attack may be confused with an acute pleuritis. The close relationship of pain to respiratory movements and the relief by splinting the chest differentiates it from herpes zoster.

XIPHOID PROCESS SYNDROME

Anterior chest and epigastric pain in this syndrome is associated with a very tender xiphoid process (32). Any body activity that imparts motion to the xiphoid process may induce local or radiating pain, and pressure over this structure often reproduces the same symptoms.

The pain is usually a dull ache which may induce nausea. It may radiate up to the neck, shoulders and arms or down over the upper abdomen. Occasionally there is a feeling of constriction in the throat. Pain is often induced or aggravated by deep breathing or movements of the trunk, such as bending and turning, as in root disease. It may occur during walking and simulate angina pectoris. When activity is restricted, symptoms usually subside, but it may be weeks or months before the pain and tenderness disappear. Recurrences are common. Infiltration of the xiphoid process with procaine gives substantial relief and tends to reduce recurrences (33, 34).

The diagnosis is based on the history, local tenderness, reproduction of symptoms on pressure over the xiphoid process and relief by procaine infiltration.

MANUBRIOSTERNAL SYNDROME

This syndrome, a counterpart of the xiphoid process syndrome, is characterized by attacks of sharp pain over or on either side of the manubrium, tenderness, and sometimes slight swelling at the manubriosternal articulation. Roentgen studies of this joint may show slight changes but more often no abnormalities are found. Pain is induced by changing position or by exercise such as walking, climbing stairs, bending forward, "straightening up" or hyperextending the spine, turning over in bed, and after such acts as coughing, sneezing or deep breathing; all of which at first suggests spinal root compression.

According to Söderström (35) the relation of pain to effort is sometimes sufficiently striking to suggest angina pectoris. This syndrome may occur in patients with generalized rheumatoid arthritis but it also appears without change in other joints. Lievre and Baumann (36), for example, reported 9 cases with such isolated involvement. Attacks of pain in 8 of their cases were precipitated by exercise involving the use of chest and shoulder girdle muscles as in lifting, and it occurred after cough or deep breathing. In some, symptoms persisted off and on for several years.

Fisher and Light (37) recently reported two interesting cases that illustrate the syndrome. In one, attacks of upper anterior chest pain followed the climbing of stairs. The pain extended 8-10 cm. on either side of the manubrium and, although diffuse and not well localized, seemed superficial rather than deep as

in angina pectoris. Straightening up, thrusting the shoulders back, hunching them forward, deep breathing and coughing caused an acute burst of pain which "froze" the patient for a moment. After resting for a short period discomfort cleared and the patient could perform all movements freely. During the attacks there was exquisite tenderness over the manubriosternal joint but between attacks there was no such tenderness in this case.

The second patient had attacks regularly on getting up from a sitting position. As he persisted in trying to straighten up he felt and heard a "click" in the upper sternum followed by complete relief and freedom of movement.

The diagnosis of this syndrome is based on the characteristic history, local tenderness, the reproduction of symptoms on pressure over the manubrium, and relief by procaine infiltration (35).

CERVICAL RIBS, COSTOCLAVICULAR COSTOSTERNAL SYNDROMES

When cervical ribs produce pressure symptoms, they are regularly referred to the shoulder and upper extremity and only occasionally to the upper anterior chest wall. Symptoms referable to the extremity, not the chest, usually predominate, and these have to be differentiated from spinal root disease and the costoclavicular compression syndrome. The diagnosis of cervical rib is established by the roentgen findings. In rare instances roentgenograms may be misleading for a fibrous band extending from the 7th cervical process to the 1st rib may produce pressure symptoms (38). In the costoclavicular syndrome there is narrowing of the costoclavicular space on the involved side and the roentgenogram will show asymmetry of the thoracic inlet.

Inflammatory reactions at the costochondral or chondrosternal junction are seen in generalized rheumatoid arthritis, metastatic invasions and after trauma or violent exercise. Involvement is accompanied by pain, local tenderness and, occasionally, localized swelling, dislocation or the slipping of a rib. It must be recognized that tenderness at the costochondral junctions is a regular occurrence in spinal root compression and this finding alone has often been erroneously attributed to such local conditions as costochondritis, fibrositis, tendoperiosteitis and pectoral myalgia (39).

MUSCLE SYNDROMES

Muscle syndromes of the chest wall may be due to local conditions or may follow trauma or excessive exercise. They are often secondary to spinal root compression. Root compression causes not only tenderness in the distribution of pain but muscle spasm. In shoulder girdle symptoms of this origin, for example, spasm of the posterior cervical muscle is one of the common findings. Involvement of other muscles such as the pectoralis major, pectoralis minor and scalenus anticus may also be secondary to root irritation. The so-called "pectoralis syndrome" is seen in markedly kyphotic subjects. The pectoralis muscles are shortened or contracted and tender. The presence of tenderness alone or so called "trigger areas" with pain referred to the shoulder (40) does not signify a myositis or a fibrositis and the fact that such areas can be relieved by traction points to a spinal origin rather than local muscle disease.

Isolated spasm and shortening of the pectoralis minor muscle with tenderness at the insertion of this muscle into the coracoid process of the scapular has been referred to as the "coracoid process syndrome" (41). Here, in addition to chest pain there is pain and numbness in the extremity. Abduction and hyperextension of the arm may be limited and induce symptoms down the arm. Because the brachial plexus, axillary artery and vein pass near the insertion of the pectoralis minor, spasm of this muscle in a patient with a kyphotic posture may compress these structures against the thoracic cage with the production of vascular as well as peripheral nerve symptoms. A similar situation holds for the scalenus anticus syndrome. Spasm of this muscle likewise may be primarily due to trauma or strain following excessive exercise, but more often, like the pectoral syndromes, it is secondary to spinal root irritation (25, 42, 43). Because of the muscle's anatomic relation to other structures spasm also produces a specific symptom pattern. Its spinal root origin can be established by the objective signs of this disorder.

UPPER GASTROINTESTINAL TRACT AND GALLBLADDER

THE ESOPHAGUS.—Functional or organic disorders of the esophagus produce pain or distress at the level of involvement or just above it. The pain may be experienced as a fullness or pressure, a burning sensation (heartburn) or a severe cramp, usually located substernally or on either side of the anterior

chest wall. The pain may radiate to the back, upper extremities or postauricular area. There is almost always a relation to the ingestion of food and with muscle spasm there may be difficulty in swallowing or a feeling or constriction due to the partial obstruction. Esophageal spasm or cardiospasm may be due to an anxiety neurosis or occur secondary to developmental abnormalities, esophageal ulcers, acute or chronic esophagitis, diaphragmatic hernia or carcinoma. In carcinoma substernal pain may be present early in the course of the disease before it is associated with difficulty in swallowing, regurgitation, bleeding or other symptoms. Extraluminal esophageal symptoms may result from mediastinal tumors, aneurysms or pericardial effusion. Disease of the esophagus is established by careful history, roentgen studies, esophagoscopy and, when indicated, biopsy.

DIAPHRAGMATIC HIATUS HERNIAS.—Hiatus hernias may be asymptomatic or produce unmistakable symptoms that mimic other conditions. Some maintain that only large hernias produce symptoms while others hold that the smaller ones are just as often responsible (44). In addition to such abdominal complaints as epigastric fullness and distress during or following eating, bloating, belching, heartburn, nausea, vomiting and regurgitation, anterior chest pain is also common (45). Ritvo (46) found that the most significant complaint in his cases was a feeling of weight or pressure under the xiphoid process. Not infrequently the pain is substernal or precordial in location and it may radiate to the left shoulder, arm and, occasionally, the fingers. Attacks come on during or shortly after eating and are relieved by hot drinks, standing erect or walking about.

The pain is sometimes precipitated by exertion, particularly after meals. The exertion involved is usually accompanied by an increase in intra-abdominal pressure such as occurs with lifting in a bent-forward position or straining with the steering wheel in parking. Attacks may also occur in recumbency and relief is then often obtained by assuming an erect position. In a careful study of the mechanism of the pain, Jones (44) concluded that overdistention of the lower end of the esophagus or the herniated portion of the stomach, with or without associated esophagitis or gastritis, is usually responsible for the substernal distress in this condition.

The demonstration of a hiatus hernia by roentgen examination does not necessarily indicate that it is the cause of symp-

toms. Angina pectoris must be excluded by careful history and exercise tests. When distress due to hiatus hernia occurs in the course of walking, the onset is more gradual, the duration longer and the relation to the degree of exertion is not close, as it is in angina pectoris. Also the chest pain in this condition is frequently accompanied by epigastric distress and belching, which are uncommon in angina pectoris. When prolonged attacks occur at rest they must be differentiated from acute coronary failure. Here again the associated abdominal symptoms are usually suggestive. Relief by drinking or by assuming an erect posture points to a diaphragmatic hernia as the cause of symptoms.

STOMACH AND DUODENUM.—Pylorospasm of biliary, duodenal or psychogenic origin causes pain and fullness in the epigastrium and sometimes a similar discomfort under the lower sternum. An acute gastritis or a gastric ulcer may do the same. Rarely the pain is referred to the upper left chest and down the arm. A duodenal ulcer only very rarely will cause pain over the lower anterior chest. Occasionally it causes pain in the interscapular region posteriorly. The presence of associated nausea and belching, the relation of symptoms to meals and the roentgen findings will establish the diagnosis.

CHOLELITHIASIS AND CHOLECYSTITIS.—In addition to right upper quadrant pain commonly referred to the right shoulder or right anterior chest, the pain in acute cholecystitis is sometimes referred to or felt over the lower substernum or precordium with radiation to the left shoulder. Examination of the abdomen will usually reveal tenderness in the right upper quadrant and palpation over the gallbladder may induce radiation of pain to the lower sternum or precordium. The attack is differentiated from myocardial infarction by the negative electrocardiographic findings and from acute coronary failure by tenderness over the gallbladder, positive roentgen findings, etc.

Mild epigastric and lower anterior chest pain are common recurring symptoms in chronic cholecystitis. Symptoms are worse soon after eating and are sometimes aggravated by exertion, in the latter respect simulating angina pectoris. Differential diagnosis is seldom difficult, for a careful history will reveal that the same symptoms occur more often at rest and are more closely related to meals than to exertion. Exercise tests are negative and the roentgen examination usually reveals evidence of gallbladder disease.

PSYCHOGENIC CHEST PAIN

Along with chest pain of coronary and of root origin, psychogenic chest pain must be reckoned with as one of the commonest forms seen in practice. Most often the characteristics of this pain as volunteered by the patient give a quick clue to its psychogenic origin. Revealing is the mild, vague type of distress, its location near the apex rather than the substernal region and its localization to a very small area, the patient often pointing to the nipple with a "right here." Equally revealing is an area of complaint too extensive to suggest specific organic disease, covering the greater part of the left anterior and lateral chest wall and sometimes reaching down to the abdomen. It may or may not be associated with pain or numbness in any part of the left upper extremity. It is frequently associated with other psychosomatic symptoms such as headache, weakness and fatigue that is more marked in the morning, often disappearing when the patient is active or preoccupied. The pain varies greatly in quality. It may be sharp, needle-like, pinching, darting; or dull, a "heaviness," a pressure or a constricting sensation. It may be fleeting and intermittent or continuous and prolonged for hours or even days. Most often it is not related to exertion, positional changes or meals, but these relationships are sometimes noted. The patient is likely to be vague as to the time of onset, and offset is usually gradual.

In contrast to the above forms of psychogenic chest pain that are easily recognized are those that closely simulate angina pectoris, acute coronary failure, root compression and other organic causes of chest pain. When the physical examination, laboratory studies and exercise tests are negative, the possibility of psychogenic factors should be given consideration. Many patients, as a result of general information or experience with the sick, become familiar with the symptoms of disease, especially heart disease, so popularly feared. Under emotional stress this information may be used in the unconscious simulation of organic illness. Careful inquiry into the emotional status at the time of the onset of symptoms will often supply clues. Psychosomatic chest pain occurs commonly under such circumstances as (1) identification with a recent death or illness, (2) following aggressive wishes or outbursts with guilt feelings toward parents, siblings, friends or associates, (3) real or invented failure situations that threaten the patient's confidence

in himself. Underlying these precipitating circumstances are the early conflicts and deep seated disturbances that are more important for treatment than for diagnosis.

Critical emotional situations in themselves are not sufficient to suggest a diagnosis nor should we misinterpret normal anxiety over organic illness. The relation of the symptoms to the emotional conflict must be unmistakable and a therapeutic response to psychotherapy may be necessary to establish this diagnosis firmly. Even when the relation of symptoms to emotional tension is established this should not becloud the possibility of the coexistence of organic disease. Today physicians are becoming more and more familiar with the role of psychogenic mechanisms in the production of pain and other symptoms. Fuller histories are being taken in order to elicit emotional information which so often has a bearing on diagnosis.

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